Southwestern

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ANNUAL MEETING, EL PASO, OCT. 20-22

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COMING MEETINGS

97th Western Cardiac Conference, Colorado Heart Association, Denver, Aug. 15-20, 1960. Category I G. P. Credit Available.

Postgraduate Course in Pediatrics, University of Colorado School of Medicine, The Stanley Hotel, Estes Park, Colorado, Sept. 1-6, 1960.

University of Texas Postgraduate Course, Turner Home, El Paso, Sept. 18, 1960.

Southwestern Medical Association, 42nd Annual Meeting, Hotel Hilton, El Paso, Oct. 20-22, 1960.

Symposium, "Medical and Biological Aspects of Energies of Space," Hilton Hotel, San Antonio, Oct. 24-26, 1960, sponsored by Air Force School of Aviation, Brooks Air Force Base, Texas. Symposium coordinator, Jack Harmon, Southwest Research Institute, Box 2296, San Antonio 6, Texas.

American Fracture Association, Annual Meeting, Mexico City, Oct. 30-Nov. 4, 1960.

Southwest Obstetrical and Gynecological Society, Annual Meeting, Riviera Hotel, Las Vegas, Nev., Nov. 7-8, 1960.

Medical Society of the United States and Mexico, Annual Meeting, Guadalajara, Nov. 8-10, and Mazatlan, Nov. 11-12, 1960.

Texas Orthopaedic Association, Galveston, Texas, April 24, 1961.

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SOUTHWESTERN MEDICINE

VOL. XLI, NO. 8 AUGUST, 1960

Santa Fe Seminar

St. Vincent's Hospital, Santa Fe
March 22, 1960

Upper Gastrointestinal Hemorrhage

Harry D. Ellis, M.D., Chairman
Seminar Summary: R. C. Derbyshire, M.D.
Presentation of Cases: Fred Soldow, M.D.

Case 1

This 41-year-old housewife was hospitalized June 23, 1954, because of weakness, epigastric pain, fainting spells, and tarry stools.

X-ray examination revealed changes suggestive of a duodenal ulcer. Two units of blood were given.

The patient was rehospitalized August 2, 1954 and Sept. 1, 1954, again with symptoms due to upper gastrointestinal bleeding. Her hemoglobin level on admission Sept. 1 was 3.6 grams. Following the administration of several units of blood, the patient was operated upon and a subtotal gastric resection was done. At operation there was no evidence of duodenal ulceration. Examination of the resected stomach revealed a hemorrhagic gastritis without ulceration. At the time of discharge from the hospital the patient's hemoglobin level was 16.3 grams.

She was rehospitalized Oct. 21, 1954, because of progressive anemia. Clinical signs of gastrointestinal bleeding had not recurred. A bone marrow examination at this time revealed erythroid aplasia and myeloid hypoplasia.

Over the next several months numerous blood transfusions were required. The white blood count ranged from 2300 to 4100 per c. mm.

A bone marrow examination May 9, 1955, revealed changes suggesting a developing monocytic leukemia. A differential white blood count at this time revealed 23 per cent monocytes. During the next 14 months the white blood count gradually rose to 90,000 per c. mm. with 90 per cent mature and immature monocytes.

Case 2

This 67-year-old reformed bartender was hospitalized July 24, 1954, for elective surgery. About 1½ years previously he had suffered a massive gastrointestinal hemorrhage. A second severe similar hemorrhage occurred one month prior to admission.

On July 30th, a portal-caval anastomosis was done. At the time of surgery ascites (3,000 ml.) was noted. The liver was cirrhotic. Postoperatively, urinary retention developed and it was necessary to perform a transurethral resection Aug. 18, 1954. Patient was discharged in fairly good condition, August 27, 1954.

On Sept. 14, 1954, he was rehospitalized in semi-coma apparently due to barbiturate intoxication. He was discharged Sept. 22, 1954, in fairly good condition.

During the next 10 months he was clinically well.

Hospitalizations were necessary July 22, 1955, August 3, 1955, and August 16, 1955, because of hepatic coma. There was no recurrence of bleeding or ascites. Death occurred August 22, 1955.

Case 3

This 40-year-old writer was hospitalized Dec. 18, 1952, following a severe gastric hemorrhage. After admission there was no further immediate bleeding. Two units of blood were given.

At 9:00 p.m., Dec. 20, 1952, the patient complained of feeling faint. He then vomited an estimated 1500 ml. of blood. In the next few hours

six units of blood were given. An emergency gastric resection was done, beginning at 12:45 a. m., Dec. 21, 1952.

Examination of the excised specimen revealed a chronic lesser curvature gastric ulcer containing an eroded artery.

The postoperative course was complicated by evisceration (1/1/53) and by a pulmonary infarct (1/10/53). The patient was discharged in fairly good condition January 24, 1953.

Case 4

This 9-year-old female child was well until February 12, 1960, when she vomited bright red blood. This occurred again three days later. In the meantime her stools were noted to be tarry. She was transfused February 16th, 17th and 19th.

The prothrombin time, platelet count, fibrinogen level, bleeding time, Lee & White coagulation time, and partial thromboplastin time were within normal limits.

On Feb. 20th, the child complained of dizziness. Her blood pressure became unobtainable. A blood transfusion was started and an emergency partial gastric resection was carried out.

At surgery the stomach was found to contain about 500 ml. of blood. No ulcers were found. The distal half of the stomach mucosa was diffusely hemorrhagic. Pathologic report on the excised stomach specimen was: Hemorrhagic Gastritis, associated with Acute Vasculitis, probably Allergic (The Vascular Changes are similar to those seen in Schoenlein-Henoch's Purpura).

No further bleeding occurred during the next 48 hours. Intermittent gastric bleeding began again Feb. 23rd. On February 27th, the child vomited an estimated 500 ml. of bright red blood. The blood pressure fell to 50/0. A transfusion of blood was begun and 200 mg. of Cortisone were given. The blood pressure slowly returned to normal.

Cortisone in smaller amounts was continued at regular intervals. No further gastric bleeding occurred.

Cortisone was discontinued March 4th, and the patient was discharged in good condition March 5, 1960. She has remained well to date.

Commentary: Dr. R. C. Derbyshire:

I would like to comment briefly on the cases which were abstracted.

As you will notice we have a rather wide variety of cases and we have all sorts of complications from the hematological to the psychiatric. The latter was not mentioned in the case of the gastric ulcer. It's somewhat embarrassing to have a patient on whom you've done a gastric resection come back with leukemia. But this was not suspected for a long time and the diagnosis was not made until several months later. The gastrectomy was done as a life saving procedure and was an example of something I would like to mention later. The reformed bartender deserves little comment, except for the fact that a successful portalcaval shunt was done on him and he lived happily in a state of euphoria for a year afterwards. He would come in periodically with ammonia intoxication which was handled with glumatic acid. How much good it did I don't know but the final solution was to put him on a non-protein diet. Whenever he would get hungry and eat steak, he would come back in a coma.

The third case is more or less clear cut, a massive gastro-intestinal hemorrhage from a gastric ulcer. One complication was not mentioned. On the second post-operative night, the patient was caught just as he was jumping out of the window. The psychiatrist—I don't think he is here tonight —told me the reason he had bled from the gastric ulcer in the first place, was that he had an unconscious desire to commit suicide. This unconscious desire became very tangible, and fortunately the nurse caught him just as he was going out the window and the psychiatrist very conveniently was out of town, so I had to take over his duties. The next day I said, "If you jump out of the window and mess up my anastomosis, I'm going to shoot you." He then behaved very well.

In the fourth case, I don't think the diagnosis, pathologically, is entirely established as Henoch-Schoenlein Purpura. This again was an operation of desperation, and I do not recommend routine gastric resections in 9 year old children. This patient, I don't believe, bled from the suture line of the anastomosis. She was free of bleeding for three days postoperatively. Then, she began to bleed massively again. As far as I can determine, cortisone is not specific for Henoch's Purpura, but it seemed to work in this case. I guess we have to give it credit.

Differential Diagnosis: Dr. Edward Goodrich:

Rather than give a formal discussion on the differential diagnosis of gastrointestinal hemorrhage,

I thought that I would perhaps go through some aspects that are less commonly thought of and some of the points of differential diagnosis.

If a patient vomits large quantities of liquid blood or clots, or passes a grossly bloody or tarry stool and simultaneously goes into shock, there is no question of there being a bleeding lesion present somewhere in the gut. If, however, a patient enters with vague complaints of fatigability, tiredness, malaise, or loss of appetite, we are much less likely to consider a bleeding lesion of the gut as the source of her anemia. Furthermore, although a guaiac test of the stool on the rectal glove is an excellent screening test for the presence of such a lesion, it is not infallible, and we are duty-bound to consider first the gut as the cause of any hypochromic microcytic anemia.

If we have confirmed the gastrointestinal tract as the source of blood loss, then we must consider the rate of loss, even before arriving at a definitive diagnosis. Is this a very slowly leaking ulcer which will respond to a good medical regime and iron therapy? Is the lesion bleeding rapidly enough to require replacement of at least part of the blood loss? Is the patient in shock, and should an artificial blood substitute be employed to maintain kidney perfusion pressure before type specific replace is effected? It is helpful to try to quantitate the loss grossly. This is probably most conveniently done by recording replacement units needed, but it is also wise to measure the quantity of the vomitus or the tarry stool, an indelicate but frequently very helpful task. Determining the rate of bleeding may take 12 to 24 hours, or it may only require a few minutes, but it is the first clinical consideration.

Having established the rate of bleeding, and having taken the necessary measures to restore and maintain the vital processes during our work-up, we must next try to establish the level at which the bleeding occurs. The only lesion found distal to the ligament of Treitz which causes hematemesis is a marginal ulcer of the jejunum following gastrojejunostomy so that the vomiting of blood always indicates a lesion proximal to the superior mesenteric artery. On the other hand, a nosebleed can be a source of occult blood in the stool, and this fact delineates our problem. The dark middle reaches of the gastrointestinal tract are most often the source of bleeding and barring surgery, our approach must be almost entirely indirect.

It is not necessary to detail here the historical aspects of the various disease which can cause GI bleeding. However, passing note should be made of what could be called the forgetful patient. This man may have been eating Tums so regularly that he does not consider their use unusual and he only admits the presence of symptoms or the taking of medication on direct, specific, and perhaps repeated questioning. In these individuals the hazard of an incomplete history is apparent. In this locale, some of us are also faced with a language barrier which may require major exertions to elicit the nuances of history in the more obscure cases of bleeding. The family history should never be neglected as it will occasionally provide a strong clue to the source of difficulty, whether it is polypopsis, cancer, ulcerative disease, or a vascular defect of a familial nature.

The specific signs by which certain disease may be recognized are numerous, but a few minutes spent looking for these signs during the physical examination may be rewarded by an unusual and accurate diagnosis. Surrounding the lips we may find the pigmented areas or freckles of the Peutz-Jeghers syndrome, the hallmark of intestinal polypopsis. Or within the mouth, on the buccal or palatine mucosa we may find telangiectatic or purpuric spots mirroring changes occurring farther down in the intestinal tract. Delay in transit of swallowed water from the mouth to the stomach over 8 or 10 seconds can indicate an inflammatory or mitotic obstructive process in the esophagus, and particular attention should be paid to the left supraclavicular lymph nodes for metastatic enlargement. We should be mindful that the characteristic epigastric tenderness of ulcerative disease of the stomach and duodenum may be an extremely good buffer.

Cirrhosis can frequently be suspected by the finding of spider angiomata, reddened palms, breast hypertrophy or absence or sparseness of hair, as well as by the more common stigmata of hepatomegally and venous dilatation in the trunk. The localized increase of naturally-found shunts around the liver in the falciform ligament may carry enough blood to create a rumble audible by stethoscope, the Cruveilhier Baumgarten syndrome.

Because of its small lumen, bleeding tumors of the small intestine, particularly toward the lower end, are prone to cause crampy, partially obstructive symptoms. To a lesser degree, this is true of

the sigmoid colon. Of course, the classical cause of silent intestinal bleeding is a tumor of the cecum, but benign and malignant tumors of the small intestine can also behave in the same fashion.

The necessity of a rectal examination can never be overemphasized.

Certain special studies may be of great help in arriving at a diagnosis of the patient bleeding from the gastrointestinal tract. Radioiodinated serum albumen and radiochromate tagged red cells are excellent methods for determining the blood volume and its changes. However, Evans blue dye or even the observation of the amount of hematocrit fall or hemodilution occurring over a period of 24 hours after the bleeding episode can give a fairly accurate index of blood loss. Direct observation of the proximal and distant parts of the guts with the esophagoscope or sigmoidoscope may provide the answer and the latter particularly is frequently indicated in lower tract bleeding before X-rays are taken. One is hard put to think of a contraindication to the use of diagnostic X-rays in the acutely bleeding patient as long as the patient is being kept out of shock and the blood loss is being matched by replacement therapy. Although the examination can be performed in bed, using portable equipment, better films and fluoroscopic observation are possible in the department. Double contrast techniques should be part of every lower bowel work-up for bleeding. Splenoportography can delineate varices.

In massive upper GI bleeding a therapeutic test with the Sengstaken-Blakemore tube is indicated if the least suspicion of varices exists. Since this is probably the best emergency treatment for bleeding varices, and since varices account for a large portion of the massive upper GI bleeders, the diagnostic use of the tube should be encouraged. This is true, even if varices are not demonstrated on X-rays.

Duodenal washings and the secretin test are described as being helpful in the diagnosis of biliary and pancreatic neoplasia. The long intestinal tube can be used to localize a bleeding site in the jejunum or ileum; but a more direct method is available. The value of a careful exploratory operation should not be underrated, even though it carries the risk of a negative result.

Laboratory data are most useful in ruling in or out the various hemorrhagic diatheses. Included are the usual bleeding and clotting studies, the capillary fragility test, and the prothrombin time. Of occasional help are the fibrinogen, the clot dissolution or fibrinolysin, partial thromboplastin, platelet, and marrow studies. Every GI bleeder should have a bromesulphalein test performed, and any level over 15 per cent retention should make the liver highly suspect, even though in a significant number of cirrhotics with upper GI bleeding, the source is an ulcer.

Although sometimes easy, the diagnosis of gastrointestinal bleeding can often be extremely difficult, taxing both the patient and the physician. Approaching the bleeding patient with a sequential program will often provide an earlier, more exact diagnosis.

Medical Management: Dr. Charles McGoey:

On a panel with three surgeons I feel a little like a Texan at the Pearly Gates. St. Peter looked at him and asked who he was. He said he was Sam Houston Jones from Texas. St. Peter looked at him sharply, shook his head sadly and said: "All right, you can come in but you're not going to like it."

I'll confine my remarks to bleeding presumed to be originating from the stomach either from a peptic ulcer, gastritis and erosions, or from a highly suspected or known duodenal ulcer.

These problems usually come to the hospital and are more apt to be seen by the general practitioner or by the internist, following a massive hematemesis, a sudden spell of weakness or they may have noticed that they passed tarry stools.

To begin with, some people are rather veterans in this game and have had more than several episodes of tarry stools and will sometimes try to persuade their physician to handle this latest episode on an outpatient basis. This is strictly a hospital problem. Some get by out of pure neglect, but it is luck rather than skill which results in a good outcome.

In addition to bed rest on admission to the hospital, they should be immediately crossmatched, for at least four pints of blood. The old rule, that if the pulse is above 100 or the blood pressure is under 100 systolic, is still a fairly good one, and I think it is a rather useful thing to take these figures when the patient is sitting up rather than reclining.

We have all been suddenly surprised by the man who has been quietly bleeding into his gut, with a

good pulse and blood pressure in prone positions, who suddenly has neither pulse nor blood pressure in the space of five minutes. Instruct the nurses to sit the patient up before they take the routine pulse and blood pressure readings. It may avert a catastrophe.

The patient should be transfused until the hemoglobin level is over 10 grams or the red count is above three million. During periods of relatively sparse nursing observation, such as nighttime, the minimum of observation period during the night of the pulse and blood pressure reading should be every 15 minutes.

For sedation, barbiturates, rather than narcotics are preferable. If nausea and vomiting are problems, parenteral Vesprin or Compazine or similar preparations are useful until the patient can retain fluids by mouth.

Presuming that the typical problem is a known gastric or duodenal ulcer, as soon as the vomiting is controlled then you start on a full and intensive ulcer regime with anticholinergics, frequent feedings with half and half or perhaps a Meulengracht type of diet.

When to X-ray these patients comes down to a question as to whether or not we know what is going on. If this is a known patient, who's been followed by the same doctor in the past, with a chronic gastric or duodenal ulcer, I think it is probably wise to defer X-ray examination for several days.

If, however, this is a brand new problem with bleeding from the upper gastrointestinal tract of uncertain origin, then I don't think anyone would be appreciably harmed by the quick trip to the X-ray Department on a stretcher and prone examination on the table by the radiologist.

From start to finish this problem should be a joint problem between a surgeon, the general practitioner or an internist. I think the best way for us non-surgical people to get our surgical conferees' dander up is to postpone the surgical consultation until we are pretty much at the end of our rope as far as medical management goes. They should be seen early after admission by the surgical staff and decisions as to when medical treatment has failed should be a joint decision between the medical man and the surgeon.

Things that have influenced our finding in this respect are 24 or 36 hours of medical management without appreciable response. If the patient is in the older age group, I think most of us are a little inclined to give up medical management sooner and consider radical surgery a little earlier.

If we know the source of bleeding is from an old sclerotic ulcer in an ancient diabetic, that too is another reason for early surgery. A suspicion that there is possibly concomitant penetration or localized perforation of the ulcer is also another reason for earlier rather than later surgical interference.

However, we have all seen many people who have defied all these rules. Not too long ago, Dr. Derbyshire and I perspired through an 84 year old woman who was a diabetic, who was in congestive heart failure and had an old sclerotic ulcer, who bled away 4 pints in 12 hours, who very neatly settled the question of surgery by telling us in her best Spanish, that nobody with a knife was going to get in the same room with her. That lady recovered very nicely in a matter of 5 days against all the rules and regulations.

Another fairly good indication for emergency gastrectomy, day or night is if the patient has taken more than 2500 cc. of blood to keep even with the game of blood loss. That, I think is a circumstance, in which all would agree indicates immediate surgery.

The main points, I would like to emphasize, are that decisions in regard to how much, how long, medical treatment should go on and when surgical interference is proper should be a joint decision between at least two physicians. The second one is for the medical people particularly to always keep the situation well in hand by being at least 1 pint ahead of this bleeding game.

Dr. Ellis:

We are very fortunate this evening to have Dr. L. H. Wilkinson with us, from Albuquerque. Dr. Wilkinson has consented to talk with us about the surgical treatment of upper gastrointestinal hemorrhage. Dr. Wilkinson.

Surgical Treatment: Dr. L. H. Wilkinson:

Although the medical management and surgical treatment of upper gastrointestinal hemorrhage are divided for purposes of discussion, these two phases of the management (in fact, all aspects of the management) of this difficult problem should be carried out simultaneously in cases where there is significant upper gastrointestinal hemorrhage.

A few weeks ago this problem was discussed at the Saturday morning surgical rounds at the Bernalillo County Indian Hospital. Present at the

meeting were representatives from the Surgical Staff of the University of Colorado Medical School, Surgical Staff of the Veterans Administration Hospital in Albuquerque and members of the medical and surgical services of the Bernalillo County Indian Hospital. We all agreed that this problem should be managed jointly from the beginning by the internist and surgeon who should see the patient together at frequent intervals. They, of course, should avail themselves of the skill and advice of the radiologist and pathologist. But beyond this, we had difficulty in agreeing on a simplified method of management which we could outline for the House Staff. I feel this difficulty was due largely to the fact that it is so difficult for anyone to accurately answer the important question, "Is the patient continuing to bleed?"

Internists and surgeons, alike, may be placed in three categories:

1) Those who feel that all patients with upper gastrointestinal hemorrhage should be operated on at once.

2) Those who postpone surgery until even the relatives are nervous about the continued loss of blood.

3) The group which includes most of us—those who advise surgery when there has been massive bleeding or there is evidence that the patient is continuing to bleed.

We consider ourselves as belonging to the latter group and have arbitrarily set a maximum of 3,000 cc. of blood (six 500 cc. units), which we will give before advising surgery. We perhaps could just as well have chosen the figure of 2500 cc. or 3500 cc.; the object in picking a figure being an attempt to avoid observing the bleeding patient until his entire blood volume has been replaced before surgical intervention. First, the estimated blood loss is determined. If this is more than 3,000 cc. the calculated amount is replaced as rapidly as is deemed safe and immediate surgery advised.

If less than 3,000 cc., then the calculated amount is given while the diagnostic tests are being performed. If there is absence of vomiting and tarry stools and repeated observations of the hematocrit, pulse, color, etc., indicate there is no further bleeding, the patient is considered to have responded to "the transfusion test". This patient is continued on medical management. However, during the next few hours or days, if there is evidence of further bleeding, surgery is advised when more than a total of 3,000 cc. are needed to restore the patient's blood volume.

We hold steadfast to the rule of not administering more than 3,000 cc. without advising surgery. However, we do not wait until 3,000 cc. of blood are necessary before advising surgery in many patients. For example, if a patient with a chronic duodenal ulcer is a borderline surgical candidate due to previous perforation or intractability or impending obstruction and bleeds almost any amount from his duodenal ulcer, he will more than likely be advised to have immediate surgery. This advice is given because it is felt that the added factor of bleeding makes him a definite surgical candidate, and there is no need in taking the risk of further hemorrhage before surgery is performed.

From the history, gastrointestinal x-ray studies, B.S.P. and other diagnostic procedures, one usually has a reasonably accurate idea of the source of the upper gastrointestinal hemorrhage. Most often the bleeding will be due to a chronic duodenal ulcer. In these cases, due to the anticipated difficulty in handling a shortened, indurated duodenum, the abdomen is opened through a right subcostal incision which is extended across the midline in a straight line to the left costal margin.[1] Bleeding esophageal varices, esophageal hiatus hernia, upper gastric lesions, and obscure lesions are approached through an upper midline incision, primarily because of better exposure of the lower esophagus. Also because of the simplicity of opening and closing the incision.

Crawford, et al.,[2] have proposed that bleeding esophageal varices be aproached from the transabdominal route. Having followed their technique in the past few cases, we too feel this approach is preferable. Briefly, the abdomen is opened through an upper midline incision with a Sengstaken or Levine tube in place in the esophagus. A long anterior gastrotomy is preformed parallel to the long axis of the stomach. The bleeding point is identified and occluded, if possible. Two soft rubber catheters are then sutured to the end of the esophageal tube and the esophageal tube then pulled cephalad until the catheters are well up into the esophagus. Traction is then maintained on the esophageal tube while outward and anterior traction is made on the catheters, thus opening the lower esophagus.

From one to three varices are usually found in the esophagus running parallel to the long axis, and usually extending down into the stomach for a

variable distance. A 000 chromic catgut suture with swedged needle is started in the lower end of each varix and continued superiorly into the esophagus for at least six cms. as a continuous interlocking suture. Often the bleeding site is at the cardioesophageal junction or in the stomach, in which case the site of bleeding is much easier to control through the transabdominal approach. This approach has the added advantage of permitting inspection of the other intra-abdominal organs and of permitting appropriate treatment, when the diagnosis of bleeding esophageal varices is erroneous.

On the first occasion in which we utilized this approach we found the bleeding varix located 5 cm. below the cardioesophageal junction on the posterior gastric wall. This would have been almost impossible to have controlled through a transthoracic opening in the esophagus.

Not infrequently the patient with an esophageal hiatus hernia develops varying degrees of ulceration in the portion of the stomach at or above the level of the diaphragm which can be the source of alarming upper gastrointestinal hemorrhage. Through an upper midline incision the stomach is reduced into the peritoneal cavity and inspected. If there is gross evidence that the lesion is malignant, esophagogastrectomy is usually necessary. In the absence of gross evidence of malignancy the hernia is repaired by aproximating the crux of the diaphragm posterior to the esophagus and the ulcer usually promptly heals. If active bleeding is present, a gastrotomy is performed and the bleeding point sutured.

Bleeding gastric neoplasms, encountered infrequently, are best treated by resection. When located near the cario-esophageal junction, esophagogastrectomy may be necessary.

In performing subtotal gastrectomy, we routinely resect two-thirds to three-fourths of the stomach without section of the vagus nerves. For gastric lesions, a gastraduodenostomy is usually done, while a post-colic gastrojejunostomy is done following resection for duodenal ulcer.

We have not combined 50 per cent gastrectomy with vagus section for the following reasons: Vagotomy combined with gastroenterostomy has not given as good long term results as adequate gastrectomy. Failure of the former is considered to be due to the fact that incomplete vagotomy has been done or regeneration has occurred. It seems logical to assume that if 50 per cent gastrectomy alone is inadequate in the treatment of duodenal ulcer, then when this procedure is combined with vagotomy, which may prove to be incomplete, the patient is then left with an inadequate gastrectomy. Also removing 20 per cent more of the stomach can probably be done more rapidly than adding vagotomy to a 50 per cent resection. However, should this procedure prove superior to the so-called "adequate gastrectomy", we hope our thinking will be sufficiently flexible to accept it.

High gastric ulcers which appear benign grossly are treated by subtotal gastrectomy combined with simple excision of the ulcer or ligation of the bleeding point. We believe this method is superior to wedge or sleeve resection due to better postoperative gastric function and the lessened chance of recurrent ulceration.

If no varices are detected and no gastric, duodenal or upper jejunal ulceration is found, the patient is usually considered to have bled from a superficial ulcerative gastritis and a subtotal gastrectomy is performed.

When a bleeding duodenal ulcer is encountered, subtotal gastrectomy is performed. No added effort is expended to resect the ulcer. If there is active bleeding from the ulcer at the time of surgery, hemostasis is obtained by figure of eight silk sutures at the site of bleeding. If the ulcer is eroding the pancreas, these are placed parallel to the long axis of the common duct. The open duodenum is then closed by inversion. If insufficient tissue is available for inversion, a number 24 Mushroom catheter is inserted into the duodenum and the proximal duodenum closed about the catheter with a purse-string suture.

If the patient's condition is critical, duodenotomy is performed with ligation of the bleeding point. A gastroenterostomy is added if the patient's condition permits. If the patient's convalescence is satisfactory, he is returned for elective gastrectomy in four to six weeks.

If the surgeon is unable to demonstrate an esophageal, gastric or proximal duodenal lesion, the distal duodenum and upper jejunum must be carefully examined. If ulceration is found in the distal duodenum or upper jejunum, this almost certainly indicates the presence of an ulcerogenic tumor[3] of the pancreas. A careful inspection and palpa-

tion of the pancreas must be done on all such cases. If a tumor of the pancreas is found and obvious metastases are present, a total gastrectomy must be done in addition to the type of tumor resection which seems advisable. If the pancreatic tumor is detected and no gross evidence of malignancy is present then a near total pancreatectomy should be performed and frozen section requested.

This is a difficult and perhaps unfair request of the pathologist, but if he feels, after careful study, that the lesion is perhaps benign, then most surgeons feel that a subtotal gastrectomy (in addition to the partial pancreatectomy already performed) is sufficient. If no tumor is found by inspection and palpation, we feel that a near total pancreatectomy and subtotal gastrectomy should be performed. These patients having, or suspected of having, an ulcerogenic tumor of the pancreas should be carefully followed postoperatively, by gastrointestinal x-ray studies, gastric analysis, and tests for occult blood in the stool. If any pancreatic tumor remains, there will be an excessive volume of gastric secretion with extremely high acid content. Also the development of gastrointestinal ulceration seems enhanced following subtotal resection.

In summary, most lesions producing upper gastrointestinal hemorrhage are best approached transabdominally. A technique for control of bleeding esophageal varices as described by Crawford has been discussed. High subtotal gastrectomy is the treatment of choice for most gastric and proximal duodenal lesions causing upper gastrointestinal hemorrhage. While less than 100 authenticated cases of ulcerogenic pancreatic tumors have been reported, this condition should nevertheless be considered in all cases of upper gastrointestinal hemorrhage due to ulceration.

Dr. Ellis:

We are going to have the summary at this point and then our discussion. Dr. Derbyshire.

Seminary Summary: Dr. R. C. Derbyshire:

Thank you, Dr. Ellis. I should like to add my welcome to Dr. Wilkinson. We all appreciate his coming up to talk to us tonight, and the fine information which he has given us. I should also like to congratulate the other speakers. I feel that I have learned much tonight. The function of the summarizer is rather easy as he doesn't have to contribute any new ideas at all. He just has to make comments on what has been said.

I should like to make a few comments on some of the points the speakers brought out. Dr. Goodrich certainly covered his subject in fine fashion, and I have very little to add. I should like to emphasize one or two points that he made.

One of these is the diagnostic use of the Sengstaken tube in bleeding from esophageal varices. I think that if this works it is a very good maneuver to employ. If it doesn't work, I do not think that one has proven very much. If the tube is in place, and the balloon is properly inflated and bleeding stops I think you have a very good presumptive diagnosis of bleeding esophageal varices. However, the tube is not foolproof. You do not always get it in the right place with the right amount of pressure. I believe after you get the tube in and the patient continues bleeding you haven't necessarily ruled out esophageal varices and I think probably Dr. Goodrich would agree with that.

I should also like to mention a couple of things in regard to diagnosis. You've probably read about the String Test. It's rather intriguing to me. I have not yet tried it and I don't know whether I will or not until there are more reports on it. Briefly, the test consists of this. A string which has radio-opaque marks on it and some buckshot on the end is given to the patient to swallow. It is followed through by x-ray. After the string is in the proper position, and this article assures me it is very easy to get it in the proper position, never requiring more than two or three hours, Fluorescein is injected intravenously.

The string is withdrawn and examined under an ultraviolet source of light. If only blood is found on the string, it means the patient has stopped bleeding. If Fluorescein and blood is found, it means the patient is still bleeding and the authors claim one can localize the source of bleeding fairly accurately by this means. It sounds intriguing.

I should also like to mention Dr. Wangensteen's refrigeration treatment. I'd like to know more about that. Dr. Wangensteen has developed a large balloon which he places in the stomach and it is connected with a refrigeration machine beside the patient's bed, with coils going to the balloon. The stomach is kept at O degrees Centigrade or maybe a bit higher than that.

The principle is that the gastric secretions are suppressed by this cooling and he claims that he has been able to control massive gastric or duo-

denal hemorrhage by this means. After the initial control of the hemorrhage, the patient is then in better condition for surgery.

I should like to say something about Dr. McGoey's remarks concerning the quiet bleeder. I think that this cannot be overemphasized. Dr. McGoey apparently has a trick he hasn't taught me yet. That is, the sitting up of the patient to take the blood pressure. I think that that is a very fine idea, and I've never seen it done. The quiet bleeder is the terrifying one, of course. When you think you have everything under control, all of a sudden, there being no sign of bleeding at all, the bottom drops out of the blood pressure. I find this rather disconcerting.

I'd like to say a little about the naso-gastric tube in the patient who has had massive hematemesis. I think that there is a little bit of disagreement among the members of the staff here. I do not see why it should be serious. I personally favor inserting a gastric tube when the patient has had a hematemesis. I think in the first place, it serves to quiet the stomach, although it may be an ordeal just to pass the tube. The stomach can be emptied if it is too full of clots. If the patient vomits while the tube in being inserted and gets rid of the clots, so much the better. Also, I think it serves as a guide to the amount of hemorrhage, and to whether or not active bleeding continues.

The joint problem, I don't think we need to mention further. I believe all cases are handled that way in this hospital. I don't think we have had anything but cooperation between the internists and surgeons.

I was most interested in Dr. Wilkinson's remarks. Dr. Wilkinson has had great experience in this field, and I should like to remind you that his mentor was Dr. Carl Williamson of Mann-Williamson Ulcer fame.

In regard to his comments on blood loss, I believe he calls himself middle of the roader. I question the criterion of the transfusion of 3000 cc. of blood. I believe we would operate a little bit sooner than that, here.

I was particularly intrigued by his description of the transesophageal, transabdominal ligation of esophageal varices. I saw this movie which was presented in Denver last year, and this looked like a rather formidable procedure to me. I am delighted to hear that Dr. Wilkinson has tried it. I have one request to make of Dr. Wilkinson. The next time you do one of these, Doctor, would you call me and let me come down and see it or scrub with you. I should certainly like to see it. Although formidable, it seems a very sensible approach to the problem.

I certainly can't disagree with Dr. Wilkinson on his views concerning hiatus hernia and ulcer. He was hoping to stir up some controversy, but I don't think he will get any from me. I think that if the diaphragmatic hernia is repaired, the ulcer will heal.

I should like to branch out at this time, and give some other methods of treatment. We've been talking mainly about gastric resection, which I personally favor. If we could regard for a few minutes, vagotomy or various operative variations, dispassionately, I think it might be a good idea. There is much disagreement still, between the gastric resectionist and the vagotomist. A lot of us get very emotional when we discuss the problem. Personally, I'm a gastric resectionist.

Weinberg of Los Angeles, recently published an article which appealed to me, and I should like to read about his next 100 cases and how they come out. He is advocating in the case of massively bleeding duodenal ulcer, merely opening the distal portion of the stomach through the pylorus into the duodenum and securing bleeding points with suture ligatures. He then does a pyloroplasty and a vagotomy. He didn't say just how easy it is for him to do this, but he considers it a great time saving procedure and his results in his first 24 cases are impressive. In his first 24 cases treated by this manner he had no deaths. All of these were serious bleeders by the criteria which we accept. No deaths in 24 consecutive cases is an accomplishment. There was only one case in which he had serious complications. This one bled postoperatively, had to be reoperated twice, and the patient finally had a gastric resection and survived.

I should also like to say a bit about the so-called blind gastrectomy. I have been a little bit disconcerted by that term. You've probably read about it. I cannot understand the rationale behind this. Possibly Dr. Wilkinson and Dr. Goodrich would say more about it, later. I see absolutely no contraindications to doing a gastrotomy. In fact, I can't see that it adds any great hazards to the operation, and I personally like to know where

the bleeding is coming from, if I possibly can find out.

To get back to some of the cases that were presented. In the literature, usually the statement is made that in about 10 per cent of the cases that are bleeding massively from the upper gastrointestinal tract, no definite ulcer can be found. One finds only so-called erosive gastritis or hemorrhagic gastritis. That is not our experience here. I have not had time to look up all of our cases but I would say that our percentage is much higher than that. These are the disappointing, frustrating cases where no ulcer can be found.

The stomach is opened and one hopefully tries to discover something in the stomach and only erosive gastritis is found. In such cases, frequently the imagination is stretched to find even that. If an erosion is found, one marks it carefully, and quickly goes to the pathologist before he has a chance to pickle the specimen and says: "Here is the ulcer—don't forget where it is." It seems to me we have had a large number of those. Possibly we shouldn't be operating on them, but the ones we've operated on have all fulfilled the criteria for emergency surgery.

I should like to bring up one more point. That is, the ideas of Dr. Stewart of Buffalo. Dr. Wilkinson did not have time for this, but I think his ideas are well worth reporting, and many of us have been following his results for a number of years.

Dr. Wilkinson will probably remember a meeting which we attended in Dallas, I think six or seven years ago. Dr. Stewart was present and he propounded some of his ideas. Dr. Stewart, I might add, advocates immediate emergency operation on all upper gastrointestinal bleeders, that is, those patients bleeding from gastric or duodenal ulcers.

He has definite criteria for the classification of an acute bleeder. The patient has to have lost over 40 per cent of his total circulating red cells before he is placed in the emergency class. Stewart's first paper on this subject appeared in 1950. It was revolutionary and caused many gasps throughout the country.

As you probably know, if these patients are treated medically the mortality is anywhere from six to fifteen per cent depending upon the age of the patient and other circumstances. When Dr. Stewart first published his series he stated that his mortality was about eleven per cent and medical mortality was twelve per cent. Everybody said, well, you haven't accomplished very much, have you? He has continued to study this throughout the years and his series has reached 193 cases.

He has a carefully trained team, and can now delegate some of the operations to the residents. They have a definite routine, with specialized personnel in the operating room, both day and night. He has as ideal a situation for dealing with these cases as anybody could have and a well trained team. Out of his first 193 cases, the mortality of the patients under 50 years of age was three per cent. In those over 50 years of age, the mortality was eighteen per cent. I believe that anyone who can report a mortaliy of three per cent in acute bleeders—and many of these are practically exsanguinated when they're taken to surgery—is doing well. I think his results are very good and certainly deserve further consideration.

(Due to a recording breakdown the discussion which followed this seminar was lost.)

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Urology Award

The American Urological Association offers an annual award of $1,000 (first prize of $500, second price $300, and third prize $200) for essays on the result of some clinical or laboratory research in Urology. Competition is limited to Urologists who have been graduated not more than ten years, and to hospital internes and residents doing research work in Urology.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Hotel Biltmore, Los Angeles, May 22-25, 1961.

For full particulars write the executive secretary, William P. Didusch, 1120 North Charles Street, Baltimore. Essays must be in his hands before December 1.

Use of Perphenazine Orally or Parenterally Among Nonpsychotic Patients

By ROBERT J. ANTOS, M.D.,* *Phoenix*

The clinical effectiveness of perphenazine,* a ring-substituted phenothiazine, has been described by numerous investigators. In addition to its use among hospitalized psychiatric patients, perphenazine is indicated for the management of tension and anxiety associated with somatic illness in ambulatory patients.[1-4] The marked antiemetic potency of this drug was described by Wang[5] after controlled comparative studies among animals and confirmed in clinical practice.[6-8] Perphenazine is available for injection intramuscularly or intravenously, in tablets, and as suppositories. The intent of the present study* was to determine optimal dosage parameters with the various routes of administration.

Methods

Nonpsychotic patients with nausea and/or anxiety symptoms arising from toxicity, pregnancy, organic disease, or psychogenic disturbance were treated with perphenazine. Injectible perphenazine was studied in fifty patients with severe emesis for whom oral medication was not feasible. Ten received an inert injection, 20 received 5 mg. perphenazine, and 20 received 10 mg. The attending physicians did not know which patients received the active drug or which dose was used. The double-blind code was broken after physicians recorded the patients' response.

Ten patients were treated with oral perphenazine for anxiety or emesis. Each patient received a placebo and single doeses of perphenazine, varying between 2 and 12 mg., at intervals during the period of study. Information about the amount of perphenazine administered each time was withheld from the patients and their attending physicians.

Trilafon suppositories were used in ten patients. A placebo was not used in this group but the patients received either one or two suppositories and variations in results were correlated with dosage.

Results

Injectible perphenazine: Of the ten patients who had received the placebo, six stated that they had been partially relieved of symptoms. Four of the 20 patients who had been given five mg. perphenazine experienced complete relief and the others were partially benefited. Only two of the 20 patients who received 10 mg. failed to experience complete relief; one patient felt very slight nausea and the other noted slight bloating.

Perphenazine orally: Six of the ten patients had a "positive" response to the placebo and the same patients also were relieved to some extent after administration of the two mg. tablet. On four mg. daily more patients experienced partial relief but none was completely relieved. When the dosage was increased to six mg. or 10 mg., there was a notable difference between the actual therapeutic effect of the drug and the expected placebo reaction. On single administrations of 12 mg. perphenazine, five patients were completely relieved of symptoms and five reported major improvement.

Perphenazine suppositories: The eight mg. suppository provided complete relief to only two patients, six were benefited to some degree, and two did not respond. Two suppositories provided substantially better results and no patient failed to obtain at least some degree of relief. Results appeared to be somewhat better if several hours elapsed between administration of the first and second suppositories. Some patients found that results were best when one suppository was used after dinner and the second before bedtime. Pregnant patients obtained substantial relief of morning emesis by using one suppository at bedtime and another early in the morning.

The results of our trials among the various groups of patients are summarized in the table.

*Director of Research, Good Samaritan Hospital.

*Perphenazine (Trilafon) was provided for this study by R. Richard McCormick, M.D. of the Medical Research Division, Schering Corporation, Bloomfield, New Jersey.

Response to Perphenazine in 70 Patients

Experimental Administration	Complete Relief	Partial Relief	Slight Relief	No Relief
INJECTIBLE PERPHENAZINE (50 PATIENTS)				
Placebo (10 patients)	—	6	—	4
5 mg. (20 patients)	4	16	—	—
10 mg. (20 patients)	18	2	—	—
PERPHENAZINE ORALLY (10 PATIENTS)				
placebo	—	6	—	4
2 mg.	—	6	—	4
4 mg.	—	8	—	2
6 mg.	2	8	—	—
10 mg.	3	7	—	—
12 mg.	5	5	—	—
PERPHENAZINE RECTALLY (10 PATIENTS)				
one 8 mg. suppository	2	5	1	2
two 8 mg. suppositories	4	5	—	—

Comment

The "placebo effect" exhibited by several patients after administration of the inert agent was not surprising; other investigators have reported an incidence of "positive" response to placebos of 50 percent in a series of psychiatric patients[9] and of 70 percent among a group of patients with gastrointestinal symptoms.[10] We believe that a valid statement about the beneficial action of a drug can be made if the response to it is unequivocally better than to a placebo among the same or matched patients. In the present study, the lower doses of perphenazine did not produce results significantly better than those produced by the placebo; when the higher doses of perphenazine were used, a pronounced therapeutic effect was observed.

Conclusions

1. The optimal therapeutic dose of perhenazine via injection is 10 to 15 mg.

2. Optimal results with oral perphenazine occur after doses of 10 to 12 mg.

3. Two suppositories of 8 mg. perphenazine provide optimal results with this route of administration.

Summary

The double-blind method was used to study injectible perphenazine in 50 patients, oral perphenazine in 10 patients, and perphenazine suppositories in 10 patients. Anxiety or emesis, irrespective of cause, was relieved rapidly in almost all patients on doses of 10 mg. orally or by injection or 16 mg. in suppositories. On lower doses the results are not significantly better than those obtainable with placebos.

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Texas Post-Graduate School to Meet Sept. 18 in El Paso

A one-day course on preoperative and postoperative treatment will be given at the Turner Home of the El Paso County Medical Society Sunday, Sept. 18, as part of the University of Texas Postgraduate School of Medicine series.

The six-hour course will include talks by Dr. Donald B. Butler and Dr. O. A. Fly, Jr. of Houston together with six El Paso physicians: Dr. Gerald H. Jordan, Dr. M. D. Thomas, Dr. Charles E. Webb, Dr. M. S. Hart, Dr. William G. Smith and Dr. John E. White.

Dr. Butler will open the session with a talk on diagnosis and treatment of small bowel obstruction and Dr. Fly will discuss large bowel obstruction.

Intestinal surgery in the poor risk patient will be the subject of a panel discussion to conclude the morning session while a panel discussion on new developments in the management of cancer of the colon will follow individual talks on the subject in the afternoon.

The course has been approved by the Texas Academy of General Practice for six hours Category I credit. Advance registration is requested. Complimentary enrollment will be granted to residents, interns and faculty members upon satisfactory completion of application blanks.

Dr. J. Leighton Green is director of the El Paso Division of the University of Texas Postgraduate School of Medicine.

Patriotism

By Russell L. Deter, M.D., *El Paso*

President, Southwestern Medical Association

The Fourth of July has come and gone. This was the day we were supposed to celebrate the founding of this great country of ours. This was the day we were supposed to wave the Stars and Stripes; be proud of our heritage; be proud of those thirteen stripes representing the original thirteen colonies that, with vision and hope and faith, discarded the yoke of oppression and tyranny and became the United States of America; proud of those thirty-seven stars which represent the states which chose to join the original thirteen to become, on the Fourth of July, 1960, the fifty United States of America joined together in a government "of the people, by the people, for the people." This was the day we should again reassert our faith in the right of man to live in dignity and freedom from oppression.

Why do I say, "we were supposed to" and "we should have"? Too many people in this country have become cynical about patriotism and "flag wavers." They think it is childish and "kid stuff." They have even passed on this feeling to their children; and, in too many instances, to their elected representatives, leaving the impression, both nationally and abroad, that the people of this country are only interested in "what's in it for me?" or, "how much can I make out of it?"

The Sawdust Trail

The time has come for all of us to hit the sawdust trail and go down to the "Amen" corner and reaffirm our faith in the Constitution of the United States and the principles of democracy. Let us stop this business of belittling our armed forces and individuals who choose it as a career. Let us honor this proud profession that has been the nucleus of the forces which have preserved the principles of democracy since the first Fourth of July.

Let us discredit the so-called "changeocrats" who are not satisfied with the time-honored and proven principles of patriotism, morality, honor, ad infinitum, but want to change everything. They back away from the fires that have warmed the hearts of nations, people, families and individuals for centuries. They are taking off in all directions, destroying or attempting to destroy that cohesive force that keeps us driving towards our ultimate goal of freedom of man from oppression and war. That cohesive force is pride in family, pride in city, state and country. Are you ashamed of being an American? Are you a "changeocrat"? Do you thing that our fate is inescapable? The Freudian ethic, as published in the Readers' Digest, says:

"All talk of social destiny and inescapable faith is simply a symptom of general unwillingness to make the effort to do and keep doing what is necessary to maintain our way of life. If man fails in the battle with the bugs, is overwhelmed in a flood of babies, runs out of top soil, smothers in smog, or if someone in the various other ways fails to solve the problems of social adaptation, it will only be because he has given up trying."

Birthright Sold

Have we sold our birthright to the "changeocrats?" Have we given up trying? Are circumstances to which we object in national government there because of your default in not voting; in not taking leadership in your precinct meetings; in not letting your elected representatives know your opinion on national and international issues? Is it because *you* have forgotten what patriotism means? Patriotism is love of country, devotion to the welfare of our country. Have we become so enamoured with ourselves, and so devoted to our own welfare that we have forgotten that how our country goes, so shall we? Let us revive patriotism, let us wave "Old Glory" proudly again. Let us take the leadership in our precinct meetings and on up to revitalization of our national government.

Have we forgotten that the reason history repeats itself is because too many were not listening the first time? Other democracies in history failed because of the complacency of its people. If our national government is not what we want it to be *have we really quit trying? HAVE YOU?*

CPC

Clinical Pathological Conference

R. E. Thomason General Hospital, El Paso

F. P. Bornstein, M.D., Editor

Case No. 1428, April 21, 1960

Presentation of case by Robert H. Thayer, M.D.

History: Dr. Nathan Kleban

Abdominal pain and constipation for three days brought a 70-year-old Latin-American woman to the hospital on March 17, 1960.

Various historians dated onset of episodes of abdominal pain back 14, 20 or 25 years, occurring about every three or four years, accompanied at times by vomiting, and not affected by intake of food. Location of the pain was right upper abdominal quadrant or epigastrium. One historian related that the pain was present off and on in the epigastrium for three years, that a hernia was repaired in 1942, and that the pain did not recur until three years before admission, since which time it had been recurring more frequently, persisted for longer periods, and was increasing in severity. Jaundice was noted some months before admission.

First admission to this hospital was January 23, 1960. Blood pressure was 210/110, later ranged from 180/90 to 90/50. Temperature reached 102 on the second day, ranged around 100 for several days, then dropped below 99. There was slight epigastric or right upper abdominal quadrant tenderness, absent umbilicus, and herniorrhaphy scar. The liver edge was felt three finger breadths below the right costal margin.

Treated With Analgesics

The patient was treated with analgesics, parenteral electrolyte solutions, tetracycline, and an anti-cholinergic drug. Except for a single complaint of cramping there was no abdominal pain after the first day and no vomiting after the second day. Treatment was for gastric ulcer.

The patient was discharged on February 6 on an antacid preparation. It was planned to repeat X-rays in four weeks and perform surgery if the ulcer had not healed.

On February 11, 15 and 29 and March 7 and 14 the patient was seen in the clinic, free of symptoms.

On March 17 the patient was brought to the emergency room. She complained of abdominal pain of increasing severity for three days, jaundice, anorexia, vomiting, distention, weakness, joint pains, weight loss, tenesmus and inability to move her bowels, despite 12 soap suds enemas.

Physical Examination

T. 100; R. 42; P. 95; B. P. 80/60.

The patient was obese. She appeared jaundiced. The heart was slightly enlarged. Deep palpation in the epigastrium evoked slight tenderness. The liver was enlarged. There was abdominal distention. There were "palpable masses."

Hospital Course

Although the patient refused an enema in the emergency room, one was given shortly after she arrived on the ward. Return solution was colored but contained no feces. Jaundice deepened, respirations became labored and blood pressure dropped. The patient went into coma and died on March 19, 1960.

Laboratory Findings (First Admission)

X-Rays: 1-23-60: Chest—Conclusion: The chest is essentially normal in appearance, slight cardiomegaly cannot be excluded. Old arthritic changes are described in the left shoulder.

1-26-60: GB and GI series: Gall bladder series showed non-visualization, probably on the basis of calculus cholecystitis; no opaque calculi were demonstrated. Changes indicative of partial obstruction of the duodenum in its third portion,

possibly related to a superior mesenteric artery syndrome are described. Re-examination for further evaluation of this segment of the intestinal tract is recommended. The radiographic appearance of the chest is within normal limits for a patient of this age.

2-2-60: Re-examination of the upper GI tract now reveals a gastric ulcer lying on the lesser curvature. There is no evidence of duodenal obstruction.

Conclusions: Gastric ulcer.

Blood Counts:

1-23-60: WBC 15,200, Hb. 14.7 gms., Ht. 48%, Segs. 82, Lymphs. 18.

1-28-60: WBC 8,500, Hb. 13.3 gms., Ht. 42%, Stabs. 3, Segs. 69, Lymphs. 25, Monos. 2.

1-30-60—WBC 7,600, Hb. 12.4 gms., Ht. 40%, Eosin. 1, Juveniles 1, Stabs. 7, Segs. 52, Lymphs. 39.

2-5-60—WBC 8,300, Hb. 13.3 gms., Ht. 41%, Eosin. 1, Stabs. 2, Segs. 65, Lymphs. 28, Monos. 4.

Prothrombin time:

1-30-60: 95%—Patient 14.5", Control 14".

Urinalyses:

1-23-60: (Catheterized) — Straw, turbid, alkaline, S.G. 1.010, Albumin and sugar, acetone, diacetic acid, negative; WBC 3-4, Ep. cells 2-3, casts, 1-2, bacteria, non-motile rods, 3+.

1-27-60: Straw, clear, alkaline, S.G. 1.006, Albumin trace, sugar negative, few bacteria:

Feces:

1-26-60: Occult blood 3+.

2-2-60—Occult blood 2+.

Serology:

1-25-60—Negative.

Chemistry:

1-23-60: Amylase—108; Glucose—118; Urea Nitrogen—9.0

1-26-60: Amylase — 22; Co_2 capacity — 20 mEq/L; Chlorides (as NaCl)—88 mEq/L; Potassium — 3.8 mEq/L; Sodium — 124 mEq/L.

127-60: Calcium—4.3 mEq/L; Co_2 capacity—18 mEq/L; Chlorides (as NaCl) — 100 mEq/L; Potassium—3.6 mEq/L; Sodium—136 mEq/L; Van den Bergh direct—.116; Indirect—.29.

2-5-60: Calcium—4.8 mEq/L; Chlorides (as NaCl)—98 mEq/L; Potassium—4.9 mEq/L; Sodium—137 mEq/L.

Urine culture and sensitivity:

1-26-60: No growth in 48 hours.

Laboratory Findings (Second Admission)

X-Rays: 3-17-60: Abdomen, flat and erect, Chest A-p supine; Conclusion: Abdomen negative for ruptured hollow viscus; chest, hypertensive and arteriosclerotic cardiovascular disease; no evidence of failure.

Blood Counts:

3-17-60: WBC, 19,600; Hb. 13.3 gms., Ht. 40%, Stabs. 2, Segs. 80, Lymphs. 28.

3-18-60: WBC 22,900; Hb. 13.3 gms., Ht. 43%, Stabs. 18, Segs. 74, Lymphs. 5, Monos. 3.

Prothrombin Time:

3-18-60: 25%—Pt. 20", Control 13".

Serology:

3-17-60: Negative.

Urinalyses:

3-17-60: Reddish-brown, hazy, acid, S.G. 1.014, Albumin 3+, sugar, trace, WBC 0-2, occ. squamous ep. cell, 0-1 fine casts, few bacteria, numerous urate crystals.

3-18-60: (Catheterized) Positive for bile, urobilinogen negative.

Chemistry:

3-17-60: Van den Bergh—2.85; Indirect—4.65.

3-18-60: Glucose — 33; Urea Nitrogen — 47 mg%; Amylase — 41; Total protein — 5.9 gm.%; Albumin—3.6; Globulin—2.3; A/G—1.6; Van den Bergh—3.85; Indirect—6.4.

3-19-60: Alkaline Phosphatase—11.1.

Thymol Turbidity:

3-18-60: 13.5 units.

P. P. D. # 1—(right arm):

3-17-60: Negative.

Cocci. right arm:

3-17-60: Negative.

Ceph. Floc.:

3-18-60: 24 hours 3+; 48 hours 4+.

Transaminase:

3-21-60: 190 units (SGOT).

Feces:

3-18-60: Negative for occult blood.

Sedimentation Rate:

3-18-60: 48 mm./60 minutes.

Electrocardiogram:

1-29-60: RBBB—atrial damage — ? LVA.

Clinical Discussion: Dr. Robert H. Thayer

First I will make a brief review of the case. This is a 70-year-old woman, Latin-American, who came to the hospital with abdominal pain,

jaundice, constipation of three days duration, she had had similar episodes of abdominal pain upper right quadrant, intermittently, for the past 25 years. In January of 1960 she was admitted to the hospital because of abdominal pain and fever. It was noted at that time that the liver was enlarged but there was no jaundice. The white count was elevated, suggesting an acute infection. Prothrombin time was 95 per cent and a serum bilirubin was normal. A chest film was essentially normal. Two GI series were done on this admission. The first one showed a normal stomach and a partial duodenal obstruction, possibly related to a superior mesenteric artery syndrome which is a condition where the branch of the mesenteric, superior mesenteric vessels presses against the third portion of the duodenum and causes symptoms of obstruction, sometimes pain and nausea. This is an intermittent condition and I suppose that she could have had this actually. It was not seen on the second GI series, but they did find a gastric ulcer. On this admission she also had occult blood in the stool but there was no anemia. She was treated for an ulcer and was seen subsequently five times in the clinic during the month of February, and from the history we must conclude that she was entirely asymptomatic during this time, and was not jaundiced. She was again admitted in March of this year with a three-day history of abdominal pain, jaundice and constipation. However, the X-rays failed to show any signs of obstruction or anything in the diaphragm. Could we now see the X-rays?

X-Ray Discussion: Dr. Vincent Ravel

The first film that we have here is a film following the oral administration of Telepaque and we see the opaque media in the large bowel and there is no evidence of concentration in the right upper quadrant and as usual when we see nonvisualization of the gall bladder, nine times out of 10 it is due to calculus cholecystitis, although we cannot demonstrate any opaque calculi. This is the first upper GI series and there is some dilatation of the duodenum and this caused the radiologist to feel this might be related to the superior mesenteric artery syndrome. Three days later we were able to clearly demonstrate a large gastric ulcer, high on the lesser curvature. The previously observed dilatation of the duodenum is now absent and again this is a further projection at three hours showing the ulcer on the lesser curvature. Those were the significant findings from the standpoint of the x-ray examination. The liver does not look particularly enlarged, the spleen may have caused a little impression on the greater curvature of the fundus but that again could be within normal limits. This survey film of the abdomen was done on the final admission. These are skin creases caused by the patient's obesity, and calcification in the iliac vessels, and here in the upright chest film, no free air under either hemidiaphragm. There is some prominence of the left ventricular segment of the cardiac silhouette.

Dr. Thayer:

There is no evidence of intestinal obstruction?

Dr. Ravel:

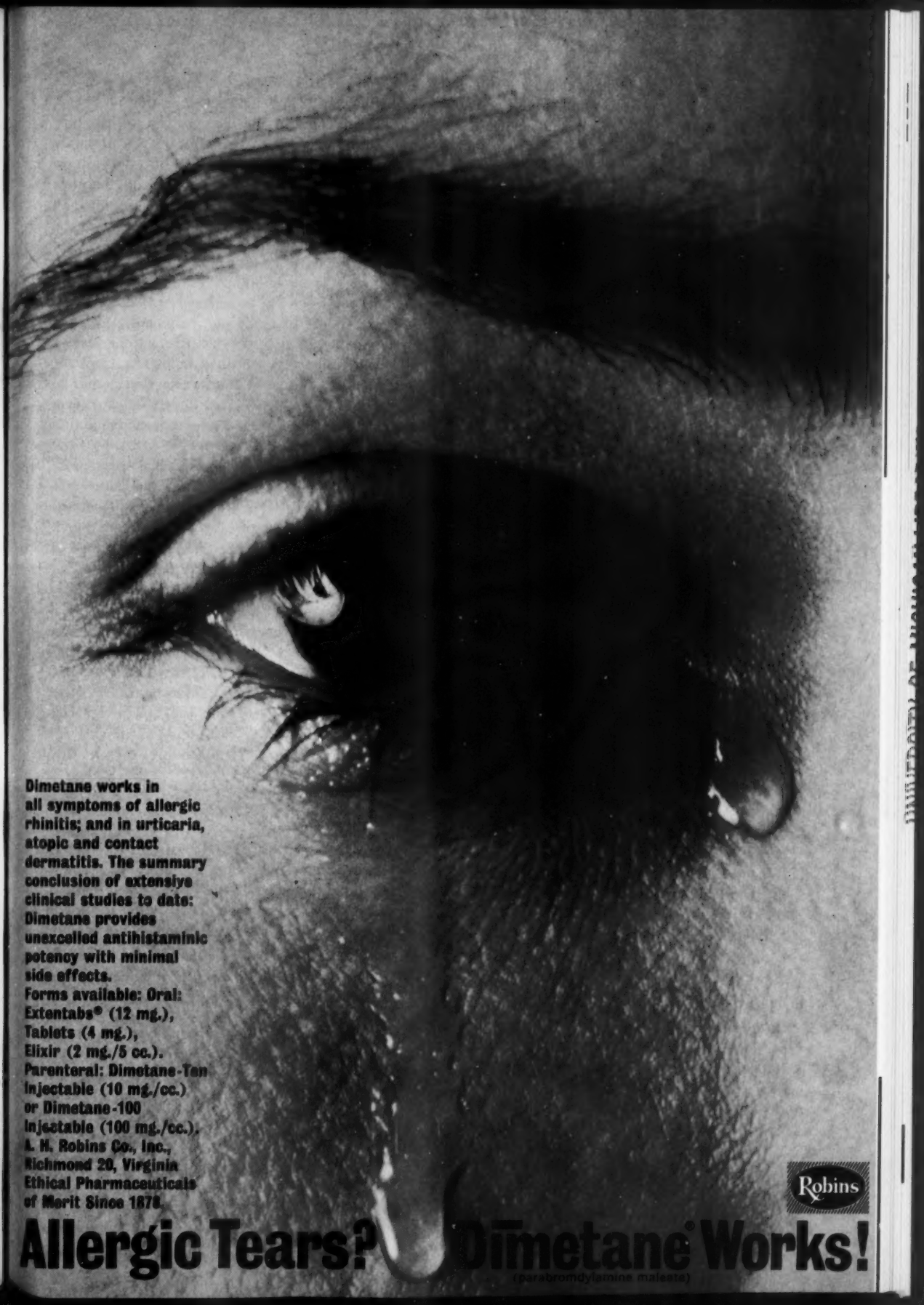
No, there is gas in the large bowel and the stomach and no evidence of ileus. The liver does not look particularly remarkable and again I cannot identify any opaque calculi, although I believe this is a case of calculus cholecystitis and possibly obstructive jaundice from cholangitis and probably hepatic failure.

Dr. Thayer:

So we have here a gastric ulcer and a gall bladder that doesn't visualize and no signs of obstruction. During her hospital stay she ran a high white count again, up to 19,000, but there was no anemia, liver function tests showed evidence of both obstructive and hepatocellular disease. Serum amylase determinations on this and on her previous hospitalization were normal. Bile was present in the urine this time, and serum transaminase was 190 which is pretty high. The stool examination was negative for occult blood on this admission and the serum proteins were slightly diminished, particularly the albumin. From the notes I gather that the patient did seem to improve and she ate all her meals on the second day and appeared to be comfortable. She had a normal colored bowel movement on the second day, which seems to be somewhat against intestinal obstruction or complete biliary tract obstruction. However, the jaundice apparently increased and on the third day she became comatose and died.

History Reconstructed

Now I have tried to reconstruct the history as best I could and the chief conclusion that I can reach is that she died in liver coma. Let us consider some of the possibilities here. Could this have been on an infectious basis? I think it is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almost certain that she had gall stones and gall bladder disease, possibly a stone in the common duct, but I don't think this was the chief cause of her death. For one thing, she didn't run much of a fever, it was a hundred when she came in and subsequently went down to normal. Furthermore, she died too quickly, she died practically in 24 hours, so I don't think that that plays a role here. There are certain vascular lesions of the liver that can produce a rapid liver death; for one, hepatic vein thrombosis is a possibility, but this usually causes severe ascites which comes on very rapidly and they don't have too much jaundice as a rule. Portal vein thrombosis can cause a very rapid portal hypertension but there is no mention in the protocol of any signs of portal hypertension.

Malignancy Involved

Could a malignancy be involved here? I think it could but here again I don't think it is the cause of her death. It is true that cancer of the colon or stomach can metastasize to the liver and cause jaundice, and we can't say that this gastric ulcer is not malignant, yet I think that her death was too rapid for this. You will recall that she seemed to be doing very well up until three days before hospitalization and then she died in another three days. A hepatoma or cancer of the Ampulla of Vater or biliary tract, the latter two particularly, could cause obstructive jaundice, yet here again I think she died too rapidly. So I intend to discount the possibility of malignancy here as the cause of death. The only thing that I can make out is that a relatively well person died quite rapidly in what appears to be hepatic coma, so I might go over some of the possibilities here. One is a viral hepatitis which can occur at any age. Some of these, though rare, will go into a post-necrotic cirrhosis and die of acute yellow atrophy quite rapidly; and this condition seems to be particularly common in post-menopausal women.

Toxic Hepatitis

The second is that of a toxic hepatitis from some drug or poison. I reviewed her records and she wasn't taking any drug that was toxic to the liver. We don't know if she was taking any outside. The third choice, and I think this is the one that I favor, is that she has had an underlying cirrhosis of the liver due to maybe alcohol or a previous case of hepatitis, and this was precipitated into liver failure by an intercurrent infection. Therefore I am going to say in this case that she had cholecystitis and a gastric ulcer that was healing and cirrhosis of the liver, rather liver failure over cirrhosis which was precipitated by probably cholecystitis but we have to think of a perforation or any tremendous insult to the liver. Another thing could have been a perforation of the gall bladder. I think the only thing I would say is she died in liver failure, probably cirrhosis.

Dr. W. R. Gaddis:

I would like to make one comment. We have had two or three other patients with a history very much like this, who had malignancies of the gall bladder. We see a number of carcinomas of the gall bladder in this territory and we never see them in the absence of stone or evidence that a stone had been present. Three years previous to her admission this patient had a non-functioning gall bladder. These patients die rather rapidly. I think this patient could well fit in the picture of malignant disease due to chronic long-standing obstruction to the biliary tree. One other thing that struck me about the record was the wide variations in blood pressure. There have been reported instances of adrenal gland tumors, pheochromocytomas, the trigger point of which was in the epigastrium. This woman could have such metastasizing pheochromocytoma with obvious malignant degeneration. I hesitate to say that is what she had on the basis of what we know, but it certainly is an interesting possibility in view of the recorded changes in the blood pressures on this patient.

Dr. Pablo Ayub:

I would like to state that the problem here was most likely an obstruction in the common duct. One thing that seems to speak against chronic illness from a cancer is that the patient was still quite obese when she came into the hospital on the final admission. I don't think that it rules it out, but I think that it makes it less likely. I think the role of obstruction which apparently from the laboratory examination is pretty positive, is probably the beginning of the end of this patient. Apparently she must have had, like Dr. Thayer said, a chronic liver ailment upon which was superimposed an obstructive type of lesion which possibly caused enough biliary cirrhosis and possibly perforation.

Clinical Diagnosis: Gastric ulcer.

Dr. Thayer's Diagnosis: Liver failure, probably cirrhosis.

Pathological Diagnoses:

1. Chronic cholecystitis with cholelithiasis, extensive neighborhood scarring, and impingement upon common hepatic duct by stone in gall bladder.
2. Jaundice.
3. Chronic inflammation and bile duct proliferation, portal spaces of liver.
4. Septic thrombosis, radicles of portal vein.
5. Focal necroses, liver.
6. 'Tubule formation', Fasciculate zone of adrenal glands.

Pathological Discussion: Dr. John Frerichs:

The patient had jaundice, and the most pertinent findings were limited to the upper abdomen. What I first encountered was an extensive zone of scarring centering primarily around the gall bladder, with contraction of the entire neighborhood so that the duodenum, part of the stomach, the hepatic flexure of the colon, even the head of the pancreas, were pulled in, as it were, all lying closer together than would have been normal. The gall bladder was small, tightly contracted over a single stone which measured 41 x 21 mm., an olive shaped stone. There was an extensive zone of scarring that ran in almost all directions and produced what, when I first started the dissection, I thought was going to turn out to be a tumor, possibly of the head of the pancreas, but as dissection went on it became obvious as scarring, all organs being intrinsically essentially normal but variously distorted and displaced, and bound around by the mass of scar tissue. The entire biliary tree, even below where this stone was, was dilated, but the stone itself had accomplished two kinds of obstruction, even though it was inside the gall bladder. The over-all sclerotic scarring around the gall bladder had bound it down so that the stone tightly over-rode the common hepatic duct, and completely obstructed it. The bile ducts above here were greatly more dilated than the distal ducts. The portal vein, also impinged upon, contained yellowish, turbid, purulent looking blood, and some of the small to middle sized intra-hepatic radicles had septic thrombus material grossly, which was not present in the main trunk.

The liver itself, specially in the central part on section grossly, had the appearance of biliary cirrhosis and a general rather tawny appearance which all turned out microscopically to be the same. I am going to have trouble explaining the stomach to at least one member of the group, namely, Dr. Ravel, since—although I knew of the X-ray finding of ulcer and looked meticulously—there was no ulcer at autopsy. The stomach being examined in the fresh condition, I was able to put the wall under considerable stretch, which will reveal any irregularities of the surface as well as thickening of the wall or ulceration, and at the time of death there was nothing abnormal. I would tend to wonder more, though, from the general relationships which are impossible to show on pictures, if the distortion and the general pulling around of the organs had not pulled this particular segment of stomach either around the edge of the liver, around the edge of this nodular scar, or in any event in some fashion to produce the appearance of an ulcer on the X-ray.

Common Duct Dilated

Here is a gross photograph which shows the bile ducts, this being duodenum, the common duct itself is dilated and here is the small gall bladder, more or less displaced, with the large stone. The gall bladder wall is reduced to a fibrous shell around this stone. The next is another view of the same thing. Here is the stone. Here the stone is removed, you can see the gall bladder lining. As noted, the entire biliary tree was dilated, and the ampulla easily passed the scissor bill which was at least three times the normal size of the ampulla. This shows the entire specimen with the gall bladder retracted; beginning about here is the take-off point of the cystic duct, so that this is the common hepatic duct and here are the right and left major ducts from the liver, disporportionately large as compared with the rest of the tract. You can also see the fixed appearance of the liver, with pigmented spots surrounded by grayish-tan connective tissue, yet the liver was not actually cirrhotic microscopically.

Portal Spaces

Microscopically one can see the portal spaces are considerably thickened with bile duct proliferation and a lot of round cell infiltration, some cells which look like fibroblasts but without collagen production, and without interruption of the liver lobulation; the lobulation is if anything more plainly visible than usual, and this is probably prior to anything that one ought to call obstruc-

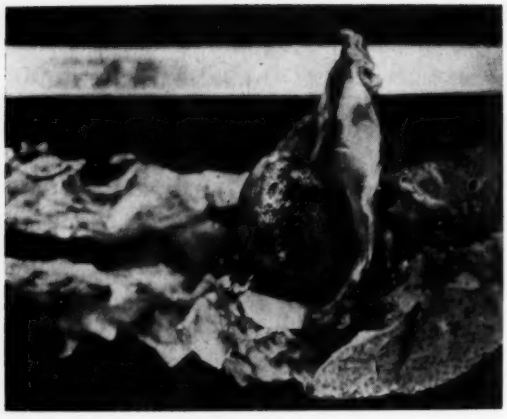

Figure 1

Gallbladder with stone, reflected upward, showing below it the area of impingement on the common hepatic duct.

tive cirrhosis, although clearly the change was going in the direction of reaching this entity. There is question about when to start using the label of cirrhosis, and personally I think that unless there is good collagen in the area, and diagonal or considerable interruption of liver lobular pattern, one should not call it cirrhosis. There was no ascitic fluid at autopsy, either. The next picture shows another low power of the liver to show one of the smaller septic thrombi in a radicle of the portal vein.

Thrombus Material

I do not know how clearly one can see that it is thrombus material but microscopically it clearly is in any event, and contains an unusually large number of leucocytes with break-down of the leucocytes and some break-down of the thrombus material itself. There were not large numbers of these in the liver but obviously enough to have caused some trouble elsewhere and not visible on a gross basis. There were numerous little foci of hepatic necrosis of which this is one, surrounded by relatively normal liver cells. In general the liver cells look healthy, but with the tiny foci of septic death, with both mononuclear and polymorphonuclear inflammatory cells, these I would relate also most likely to septic thrombosis of the portal vein. While these are fairly numerous, they are not enough to be directly responsible for her death.

The lungs did not show any pneumonia. Despite the excruciating history of attempts to get fecal material out of the patient, the intestine was loaded with it at autopsy, with hard, formed feces even in the cecum and all around the ascending, transverse and most of the descending colon. It was totally acolic stool. There was also some little hemorrhagic areas in the intestinal mucosa without ulceration. The endometrium was bloody as well, perhaps indicative of an early hemorrhagic diathesis incidental to her liver lesion.

Adrenal Gland

Here at last is the adrenal gland, and herewith the reason she died in the fashion and at the time she did. If you regard something like this as a normal fasciculate zone cell cord, the contrast between it and such structures as these, which are tubular or pseudo-tubular, is startling. What happens in the process is that the cord itself develops a lumen in its middle, where normally there is none; a little serous fluid is sometimes visible in this, as here, and occasionally inflammatory cells or necrotic cortical cells desquamate into the lumen of the tubules. This patient had a moderately bad case of this change, by no means as bad as

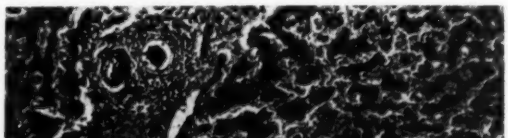

Figure 2

General pattern of liver.

they come, but on the other hand any definitely detectable degree of this change is an important finding, and ordinarily enough is taken to account for death.

This is the sort of lesion which has been described off and on for the last 50 years, most recently by Rich, in the early 1940's, and then

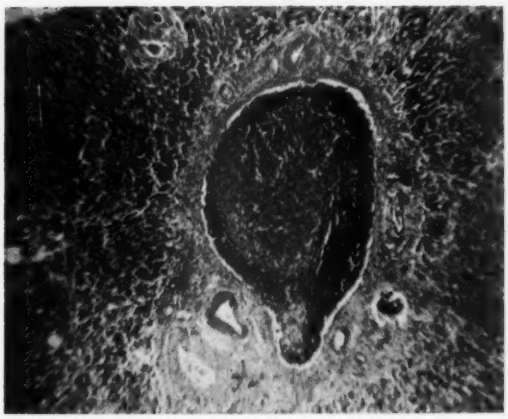

Figure 3

One of the thrombi in radicle of portal vein.

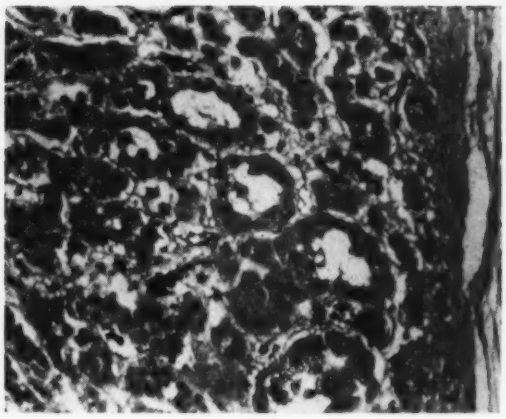

Figure 4

'Tubules' in the adrenal fasciculate zone.

relatively understandable—which it had not earlier been—due to the growing knowledge of adrenal cortical function. The change co-related with 'septic' deaths in which there was marked circulatory collapse, as appears to have been the case here, since the last recorded blood pressure is very low. The cause of the change is not clearly known, but it may come from endogenous ACTH since a change similar to it can be produced experimentally with ACTH. In terms of natural human disease it occurs with one or another kind of overwhelming infectious situation, and it does correlate with shock and sometimes with sudden death, or at least with rapid death. In this case it appears that the septic thrombosis of the portal vein and highly probably septicemia lead to the 'tubule' formation, and this was surely an adequate cause of the severe circulatory collapse, which immediately lead to death.

Dr. Kleban:

Is that associated with shock, not associated with infection?

Dr. Frerichs:

No, it seems not to be associated with pure shock, or such situations as crush syndrome, etc., in the absence of infection.

Dr. Ravel:

I note an interval of about two months between the films and death, and healing of the ulcer could easily take place in that time; I have seen them heal in 8 days.

Dr. Frerichs:

All I can tell you is I couldn't find it, or any evidence of scarring.

Dr. Kleban:

I think that as far as learning something from this patient is concerned that there is one single outstanding thing. The boys who saw this patient on her first hospital admission ignored their own history, their own physical findings, and presumably had insufficient confidence in themselves to accept the diagnosis of the radiologist. They knew that she had an enlarged liver. Well, I don't think it was important whether it was the liver or whether it was the scarring that the recurrent cholecystitis had caused, but once they got a diagnosis of gastric ulcer from the radiologist, that was their diagnosis and they thereafter paid no attention to their own findings. We all of us I think tend to do that, but they lacked confidence presumably in their own history and their own physical and accepted the radiologist's diagnosis, which proved to be a fatal mistake for this lady.

Dr. Frerichs:

I think your comment is pointed, since among other things, ulcer or no ulcer, it would hardly account for the jaundice, which was noted on the history as having existed in the past and jaundice is usaully a reasonably worthwhile symptom to look into even if it has disappeared by the time one sees the patient.

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